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☐	L1	(pituitary adenylate cyclase activating polypeptide) and @pd > 20040113	6

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           changes

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=> s pituitary adenylate cyclase activating polypeptide or PACAP
L1       6494 PITUITARY ADENYLATE CYCLASE ACTIVATING POLYPEPTIDE
OR PACAP

=> s l1 and (knockout or transgen? or disrupt? or delet? or deficien?)
L2        366 L1 AND (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR DELET?
OR DEFICIEN?
                )

=> d bib abs

L2  ANSWER 1 OF 366  BIOSIS  COPYRIGHT 2004 BIOLOGICAL ABSTRACTS
INC. on STN
AN  2004:13351  BIOSIS
DN  PREV200400012847
TI  ***PACAP*** treatment maintains the beta cell mass and retards the
    onset of hyperglycemia in beta cell-specific calmodulin-overexpressing
     ***transgenic*** mice.
AU  Tsunekawa, S. [Reprint Author]; Miura, Y. [Reprint Author]; Yamamoto, N.
    [Reprint Author]; Ariyoshi, Y. [Reprint Author]; Senda, T.; Oiso, Y.
    [Reprint Author]; Niki, I.
CS  First Dept of Internal Medicine, Nagoya University, School of Medicine,
    Nagoya, Japan
SO  Diabetes & Metabolism, (August 2003) Vol. 29, No. Hors serie 2, pp. 4S58.
    print.
    Meeting Info.: 18th International Diabetes Federation Congress. Paris,
    France. August 24-29, 2003.
    ISSN: 1262-3636.
DT  Conference; (Meeting)
    Conference; Abstract; (Meeting Abstract)
LA  English
ED  Entered STN: 24 Dec 2003
    Last Updated on STN: 24 Dec 2003


=> d his

    (FILE 'HOME' ENTERED AT 16:51:12 ON 05 FEB 2004)

    FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 16:51:20 ON 05 FEB 2004
L1       6494 S PITUITARY ADENYLATE CYCLASE ACTIVATING POLYPEPTIDE
OR PACAP
L2        366 S L1 AND (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR
DELET? OR DEFICI

=> s l1 (1s) (knockout or transgen? or disrupt? or delet? or deficien?)
L3        280 L1 (1S) (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR DELET?
OR DEFICIEN
             ?)

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4        154 DUP REM L3 (126 DUPLICATES REMOVED)

=> s l4 and PY=2002
L5         35 L4 AND PY=2002

=> s l4 and PY<=2002
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L6        124 L4 AND PY<=2002

=> d his

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L1       6494 S PITUITARY ADENYLATE CYCLASE ACTIVATING POLYPEPTIDE
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L2        366 S L1 AND (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR
DELET? OR DEFICI
L3        280 S L1 (1S) (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR
DELET? OR DEFIC
L4        154 DUP REM L3 (126 DUPLICATES REMOVED)
L5         35 S L4 AND PY=2002
L6        124 S L4 AND PY<=2002

=> s l2 and psychiatr?
L7          3 L2 AND PSYCHIATR?

=> dup rem l7
PROCESSING COMPLETED FOR L7
L8          3 DUP REM L7 (0 DUPLICATES REMOVED)

=> d bib abs 1-
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L8  ANSWER 1 OF 3  CAPLUS  COPYRIGHT 2004 ACS on STN
AN  2003:1004671  CAPLUS
DN  140:3795
TI  Non-human animal model for  ***psychiatric***  disorder with
     ***deficient***  in function of  ***pituitary***  ***adenylate***
     ***cyclase***  -  ***activating***  ***polypeptide***  gene
IN  Baba, Akemichi; Matsuda, Toshio; Hashimoto, Hitoshi; Shintani, Norihito
PA  Japan
SO  U.S. Pat. Appl. Publ., 13 pp., Cont.-in-part of U.S. Pat. Appl. 2001
    34,885.
    CODEN: USXXCO
DT  Patent
LA  English
```

FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2002162128	A1	20021031	US 2002-73135	20020213
US 2001034885	A1	20011025	US 2001-835627	20010417
PRAI JP 2000-118088	A	20000419		
US 2001-835627	B2	20010417		

AB The invention relates to mammalian model animal for ***psychiatric*** disorders having a chromosome of a somatic cell and a germ cell with ***deficiency*** of function of ***pituitary*** ***adenylate*** ***cyclase*** - ***activating*** ***polypeptide*** (***PACAP***) gene. The exon 5 of gene ***PACAP*** of mammal is ***disrupted*** and replaced with neomycin resistance gene. The results of behavioral expts. with ***PACAP*** -/- mice demonstrate that ***disruption*** of the ***PACAP*** gene in mice lead to perturbations in psychomotor behaviors, esp. the exploratory component of locomotor behavior, implicating ***PACAP*** in psychotic brain functions. Furthermore, the 5-HIAA level was decreased slightly in the cortex and striatum of the ***PACAP*** -/- mouse brain. One of the striking findings of the present study was that ***PACAP*** -/- mice showed abnormal jumping behavior in the open field arena. The ***PACAP*** -/- mouse should be a valuable tool to investigate both normal and pathol. processes in which ***PACAP*** has been proposed to play a role.

L8 ANSWER 2 OF 3 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2002:587826 BIOSIS
DN PREV200200587826
TI Higher brain functions of ***PACAP*** and a homologous Drosophila memory gene amnesiac: Insights from knockouts and mutants.
AU Hashimoto, Hitoshi; Shintani, Norihito; Baba, Akemichi [Reprint author]
CS Laboratory of Molecular Neuropharmacology, Graduate School of Pharmaceutical Sciences, Osaka University, 1-6 Yamadaoka, Suita, Osaka, 565-0871, Japan
baba@phs.osaka-u.ac.jp
SO Biochemical and Biophysical Research Communications, (September 27, 2002)
Vol. 297, No. 3, pp. 427-432. print.
CODEN: BBRCA9. ISSN: 0006-291X.
DT Article
LA English
ED Entered STN: 13 Nov 2002
Last Updated on STN: 13 Nov 2002
AB Neuropeptides usually exert a long-lived modulatory effect on the small-molecule neurotransmitters with which they colocalize via regulation of the response times of second messenger systems. ***Pituitary*** ***adenylate*** ***cyclase*** - ***activating*** ***polypeptide*** (***PACAP***) functions as a neuromodulator and neurotransmitter and regulates a variety of physiological processes. ***PACAP*** is structurally highly conserved during evolution, implying its vital importance. In Drosophila, loss-of-function mutations in a ***PACAP*** -like neuropeptide gene, amnesiac (amn), affect both memory retention and ethanol sensitivity. The amnesiac gene is expressed in neurons innervating the mushroom body lobes, the olfactory associative learning center. Conditional genetic ablation of neurotransmitter release from these neurons mimics the amnesiac memory phenotypes, suggesting an acute role for amnesiac in memory. However, genetic rescue experiments also suggest developmental defects in amnesiac mutants, implying a role in neuronal development. There is a parallel between memory formation in Drosophila and mammals. ***PACAP*** -specific (PAC1) receptor- ***deficient*** mice show a deficit in hippocampus-dependent associative learning and mossy fiber long-term potentiation (LTP). Meanwhile, ***PACAP*** - ***deficient*** mice display a high early mortality rate and additional CNS phenotypes including behavioral and psychological phenotypes (e.g., hyperlocomotion, intense novelty-seeking behavior, and explosive jumping). A functional comparison between ***PACAP*** and amnesiac underlines phylogenetically conserved functions across phyla and may provide insights into the possible mechanisms of action and evolution of this neuropeptidergic system.

L8 ANSWER 3 OF 3 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1999:28142 BIOSIS
DN PREV199900028142
TI Truncated presenilin 2 derived from differentially spliced mRNAs does not affect the ratio of amyloid beta-peptide 1-42/1-40.
AU Gruenberg, Juergen; Walter, Jochen; Eckman, Chris; Capell, Anja; Schindzielorz, Alice; Younkin, Steven; Mehta, Nitin; Hardy, John; Haass, Christian [Reprint author]
CS Central Inst. Mental Health, Dep. Molecular Biol., J5, 68159 Mannheim, Germany
SO Neuroreport, (Oct. 5, 1998) Vol. 9, No. 14, pp. 3293-3299. print.
CODEN: NERPEZ. ISSN: 0959-4965.
DT Article
LA English
ED Entered STN: 3 Feb 1999
Last Updated on STN: 3 Feb 1999
AB Numerous mutations in the presenilin (PS) genes cause early onset familial Alzheimer's disease (FAD). Here we characterize the expression of two naturally occurring alternative PS2 transcripts which lack either exons 3 and 4 (PS2 DELTAexon3,4) or exons 3, 4, and 8 (PS2 DELTAexon3,4,8). These transcripts do not contain the natural initiation codon within exon 3. The transcripts are efficiently translated as N-terminal truncated proteins. These ***deleted*** proteins are still able to regulate formation of endogenous PS fragments, indicating that the C-terminal half of the PS2-protein is sufficient for this phenomenon. Although appx50% of the PS1 and both PS2 mutations occur within the N-terminal region lacking in the PS2 DELTAexon3,4 and PS2 DELTAexon3,4,8 proteins, expression of these truncated proteins does not affect pathological generation of amyloid beta-peptide (Abeta). This suggests that point mutations causing AD are gain of function mutations.

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FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 16:51:20 ON 05 FEB 2004
L1 6494 S PITUITARY ADENYLATE CYCLASE ACTIVATING POLYPEPTIDE OR PACAP
L2 366 S L1 AND (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR DELET? OR DEFICI
L3 280 S L1 (1S) (KNOCKOUT OR TRANSGEN? OR DISRUPT? OR DELET? OR DEFIC
L4 154 DUP REM L3 (126 DUPLICATES REMOVED)
L5 35 S L4 AND PY=2002
L6 124 S L4 AND PY<=2002
L7 3 S L2 AND PSYCHIATR?
L8 3 DUP REM L7 (0 DUPLICATES REMOVED)

=> s l2 and homozygo?
L9 7 L2 AND HOMOZYGO?

=> dup rem l9
PROCESSING COMPLETED FOR L9
L10 4 DUP REM L9 (3 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 4 ANSWERS - CONTINUE? Y/(N):y

L10 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:282277 CAPLUS
DN 138:282471
TI Use of human and mouse insulin 6 gene-encoded protein in improving spermatocyte motility in diagnosis and treatment of male sterility
IN Menon, Ram K.; Sperling, Mark A.; Lu, Chunxia; Witchel, Selma; Kasik, John
PA Children's Hospital of Pittsburgh, USA
SO PCT Int. Appl., 92 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003028457	A1	20030410	WO 2002-US30781	20020927
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2003229035	A1	20031211	US 2001-967399	20010928
PRAI US 2001-967399	A	20010928		

AB The present invention relates to a novel gene from the insulin family, INSL6, which expresses a protein restoring motility in ciliated cells. The proteins of the insulin family play essential roles in pleiotropic physiol. processes affecting metab., growth, and reprodn. A new member of the insulin family named Ins16 is disclosed playing an essential role in ciliated cell activity. Ins16 plays an essential role in spermatocyte function. Thus, the INSL6 gene and its protein product are useful in the treatment of infertility caused by the loss of spermatocyte motility. A method of modulating male fertility is disclosed.
RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 2 OF 4 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:504687 BIOSIS
DN PREV200100504687
TI Sympathoadrenal function in ***pituitary*** ***adenylate*** ***cyclase*** - ***activating*** ***polypeptide*** (***PACAP***)- ***deficient*** mice.
AU Hamelink, C. R. [Reprint author]; Lee, H. W. [Reprint author]; Damadzic, R. [Reprint author]; Tjurmina, O.; Young, W. S. [Reprint author]; Weihe, E.; Eiden, L. E. [Reprint author]
CS Lab. of Cellular and Molecular Regulation, NIMH, NIH, Bethesda, MD, USA
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 620. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience. San Diego, California, USA. November 10-15, 2001.
ISSN: 0190-5295.

DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 31 Oct 2001
Last Updated on STN: 23 Feb 2002
AB ***PACAP*** 's role as a splanchnic neurotransmitter regulating adrenomedullary secretion is imprecisely defined. We generated ES cells heterozygous for ***PACAP*** ***deletion*** by homologous recombination, and from them, mice ***homozygous*** for the wild-type (+/+) or null (-/-) ***PACAP*** allele. Challenge with 2-5 U/kg of insulin resulted in decreased survival, a less profound elevation of circulating epinephrine, and a more profound hypoglycemia, in (-/-) than in (+/+) mice. Decreased survival of (-/-) mice after insulin challenge could be partially reversed by concommitant administration of glucose (20ug/mouse/hour, i.p.), isoproterenol (3ug/mouse/hour, i.p.), or ***PACAP*** (10nmol/mouse single dose, i.p.) with 5 U/kg insulin (i.p.). In addition to decreased epinephrine output in ***PACAP*** (-/-) mice following insulin, ***PACAP*** (-/-) mice exhibited no elevation in the activity of adrenal tyrosine hydroxylase, the rate-limiting enzyme in catecholamine biosynthesis, whereas adrenal tyrosine hydroxylase activity was doubled 4-8 hours after insulin administration (2 U/kg) in ***PACAP*** (+/+) mice. These data suggest that ***PACAP*** is required to couple secretion and biosynthesis of adrenomedullary catecholamines to maintain plasma catecholamine levels sufficient for gluconeogenesis during prolonged hypoglycemia.

L10 ANSWER 3 OF 4 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 1
AN 2000:491720 BIOSIS
DN PREV200000491841
TI ***Pituitary*** ***adenylate*** ***cyclase*** - ***activating*** ***polypeptide*** precursor is processed solely by prohormone convertase 4 in the gonads.
AU Li, Min [Reprint author]; Mbikay, Majambu; Arimura, Akira
CS U.S.-Japan Biomedical Research Laboratories, Tulane University Hebert Center, 3705 Main Street, Belle Chasse, LA, 70037-3001, USA
SO Endocrinology, (October, 2000) Vol. 141, No. 10, pp. 3723-3730. print.
CODEN: ENDOAO. ISSN: 0013-7227.
DT Article
LA English
ED Entered STN: 15 Nov 2000
Last Updated on STN: 10 Jan 2002
AB ***Pituitary*** ***adenylate*** ***cyclase*** - ***activating*** ***polypeptide*** (***PACAP***) is abundant not only in the brain, but also in the testis. Immunohistochemical studies have shown that ***PACAP*** -LI in rat testis is expressed stage specifically in spermatids. This suggests that testicular ***PACAP*** participates in the regulatory mechanism of spermatogenesis. Additionally, the ovary contains a relatively small amount of ***PACAP*** , conceivably involved in the regulation of folliculogenesis. ***PACAP*** is synthesized as a preprohormone and is processed by prohormone convertases, such as PC1, PC2, and PC4. PC4 is expressed only in the testis and ovary, where neither PC1 nor PC2 is expressed. However, whether PC4 is the sole endoprotease for the ***PACAP*** precursor in the gonads remains unknown. Recent studies using PC4- ***transgenic*** mice revealed that male PC4-null mice exhibited severely impaired fertility, although spermatogenesis appeared to be normal. The female PC4-null mice exhibited delayed folliculogenesis in the ovaries. To examine whether PC4 is the sole processing enzyme for the ***PACAP*** precursor in the gonads, we analyzed testicular and ovarian extracts from the PC4-null and wild-type mice for ***PACAP*** (PACAP38 and PACAP27) and its messenger RNA using reverse phase HPLC combined with specific RIAs and ribonuclease protection assay, respectively. For RIAs, three different polyclonal antisera with different recognition sites were used to identify PACAP38, PACAP27, and its precursor. Neither the testis nor the ovary from the PC4-null mice expressed PACAP38 or PACAP27, but the levels of ***PACAP*** transcripts in the testis and ovary of ***homozygous*** PC4- ***deficient*** mice were considerably elevated compared with those of the wild-type and heterozygous animals. The findings indicate that PC4 is the sole processing enzyme for the precursor of ***PACAP*** in the testis and ovary of mice. The possibility that the absence of bioactive ***PACAP*** in the testis and ovary of PC4-null mice caused severely impaired fertility in the males and delayed folliculogenesis in females warrants investigation.

L10 ANSWER 4 OF 4 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 2
AN 1995:34746 BIOSIS
DN PREV199598049046
TI Molecular Basis of Familial Growth Hormone ***Deficiency*** .
AU Perez Jurado, L. A.; Argente, J. [Reprint author]
CS Div. Paediatr. Endocrinol., Hosp. Nino Jesus, Avda. Menendez y Pelayo, 65, E-28009 Madrid, Spain
SO Hormone Research (Basel), (1994) Vol. 42, No. 4-5, pp. 189-197.
CODEN: HRMRA3. ISSN: 0301-0163.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 25 Jan 1995
Last Updated on STN: 14 Mar 1995
AB A significant proportion of cases of GH ***deficiency*** (5-30%) may be due to genetic causes. At least four Mendelian types of isolated GH ***deficiency*** (IGHD) have been delineated based on the mode of inheritance and the degree of GH ***deficiency*** : IGHD type IA, autosomal recessive with absent endogenous GH; type IB, autosomal recessive with diminished GH; type II, autosomal dominant with diminished GH; and type III, X-linked with diminished GH. Most patients with IGHD type IA have heterogeneous ***deletions*** , ranging in size from 6.7 kb to 45 kb, that encompass the entire gene encoding for pituitary GH, GH-1. Nonsense, frameshift and splice GH-1 mutations that predict a complete lack of bioactive GH synthesis in ***homozygotes*** have also been reported in association with IGHD IA. Additionally, some cases of IGHD type II have dominant negative mutations in one allele of the GH-1 gene. Panhypopituitary Dwarfism (PD), a condition characterized by ***deficiency*** of at least other pituitary trophic hormone in addition to GH ***deficiency*** , can have autosomal and X-linked modes of inheritance. Interestingly, both recessive and dominant mutations at the gene encoding for the pituitary transcription factor Pit-1 have been found in a specific subtype of PD that combines GH, prolactin and TSH ***deficiencies*** . In contrast, the loci and mutations responsible for the other Mendelian forms of IGHD and PD remain unknown. Linkage studies using genetic markers have excluded the GH locus on chromosome 17 in apprx 50% of the cases and the GH-releasing hormone (GHRH) locus on chromosome
20 in all the studied families (types IB and II) in whom the mutation cannot be traced to defects in these genes. Furthermore, several uncharacterized loci on the X chromosome must be required for normal GH secretion. In summary, genetic studies have provided a better understanding of the mechanism of GH ***deficiency*** as well as new tools for specific diagnosis of several forms of IGHD and PD. However, isolation and evaluation of other genes involved in GH secretion is still necessary. Several possible candidate genes have been recently cloned and characterized, including genes encoding the human GHRH receptor, the ***pituitary*** ***adenylate*** ***cyclase*** ***activating*** ***polypeptide*** (***PACAP***) and the ***PACAP*** receptor. Analysis of these genes in IGHD and PD families may clarify the molecular basis of the defect and also provide new insights into the complex regulation of GH.

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FULL ESTIMATED COST		0.06	69.34

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) TOTAL	ENTRY	SINCE FILE SESSION	
CA SUBSCRIBER PRICE		0.00	-1.39

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